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2	295	((fly or Drosophila) with (dpp or decapentaplegic))	USPAT; US-PGPUB; EPO; DERWENT	2003/04/03 06:52
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Bone morphogenetic proteins induce apoptosis and growth factor dependence of cultured sympathoadrenal progenitor cells.

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Neuron numbers in developing vertebrate organisms are regulated by the availability of growth factors which promote their survival. However, neuron survival may also be regulated by growth factors which promote rather than prevent cell death. This study examined the effects of bone morphogenetic proteins (BMPs) in inducing apoptosis of MAH cells, an immortalized sympathoadrenal progenitor cell line. Treatment of MAH cells with BMP2 or BMP4 killed the cells in a dose-dependent manner. By contrast, treatment with BMP7 or TGFbeta1 failed to affect survival, suggesting that induction of apoptosis is specific to the dpp subgroup of BMPs. Survival after treatment with BMP2 or BMP4 required addition of fibroblast growth factor (FGF) and nerve growth factor (NGF), indicating that BMP treatment made the neurons dependent upon an exogenous factor for survival. Several experimental observations suggested an apoptotic mechanism for BMP-induced death. After BMP2 treatment, the cells progressively shrank and became pyknotic. Further, there was prominent endonucleosomic cleavage of DNA (laddering) as well as TUNEL staining. Moreover, BMP-induced death was inhibited by the caspase inhibitor z-VAD and was partially prevented by the endonuclease inhibitor aurintricarboxylic acid. These observations suggest that neuron numbers may be regulated by factors which promote death and that exposure to such factors may be a signal for the development of dependence upon other growth factors for survival.

PMID: 9527885 [PubMed - indexed for MEDLINE]

1: Dev Biol 1997 Jun 15;186(2):127-38 Related Articles, Books, LinkOut

Msx2 is a transcriptional regulator in the BMP4-mediated programmed cell death pathway.

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Homeobox-containing genes play an important role in patterning processes that occur during embryogenesis. Programmed cell death is a key process during pattern formation. The mechanisms by which programmed cell death is spatially regulated are not well characterized. Msx1 and Msx2 are two closely related homeobox-containing genes that are expressed at sites where cellular proliferation and programmed cell death occur, including the developing limb and the cephalic neural crest. Tissue interactions are necessary for the maintenance of Msx1 and Msx2 expression and programmed cell death. It has been demonstrated that BMP4 can regulate cell death at these same sites as well as induce Msx expression. These observations lead to the hypothesis that Msx2 is a key regulator of cell death in the BMP-mediated pathway. Embryonic stem cell lines will undergo processes typical of early embryogenesis upon aggregation and have recently been shown to provide a model system for programmed cell death. In contrast to ES cells, we see that P19 cells do not undergo pronounced cell death upon aggregation; however, constitutive ectopic Msx2 expression in P19 cells results in a marked increase in apoptosis induced upon aggregation but has no effect when cells are grown as a monolayer. If aggregates are allowed to interact with a substrate, the process of programmed cell death is completely inhibited. Addition of BMP4 to aggregated P19 cells also results in cell death; however, BMP4 does not increase levels of cell death in Msx2-expressing cells. Addition of BMP4 to P19 cells results in an induction of Msx2 transcription consistent with its proposed role in cell death in the embryo. Our data support a model by which BMP4 induces programmed cell death via an Msx2-mediated pathway and provide direct functional evidence that Msx2 expression is a regulator of this process.

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